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VERTIGO.*

DR. JOHN A. MALCOLM, Pittsburgh.

Dizziness is one of the most common symptoms offered by patients. While it is often a secondary symptom, it is, in certain cases, the chief complaint.

Very early in clinical practice it is clear that patients' conceptions of dizziness are by no means the same. Since it is a subjective symptom, we are dependent on the patient's observation and ability to describe his feelings. Often the history is that of a previous episode, so that memory must be prodded to gain certain facts. The time taken to analyze the complaint and to classify it accordingly will serve to give a clearer picture. What is called dizziness may prove to be a faintness, giddiness, unsteadiness, blurring of vision, impairment of consciousness or a strange feeling in the head. Some persons may even refer the dizziness to some part of the body, such as the stomach.

For our present discussion we must limit dizziness to what we understand as true vertigo. As the most essential characteristic it must include a sense of motion. This may be a rocking sensation or a linear motion in various directions, but it is most often rotary. It is called subjective if the body seems to be turning and objective if the surroundings seem to be turning, but the relative effect is the same. A rotation to right or left in the long axis of the body is usual, although it may be an impression of turning end over end. Such sensations connote disturbed balance if the patient is upright, but whatever his position the effect is a disorientation in space from a mild to an extreme degree. If severe, the recum-

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bent patient clutches for anchorage in fear of falling from the bed. Secondary symptoms, such as nausea or vomiting, need not be present but are indicative of the acuity and severity of the vertigo. In milder degrees the patient can walk but is uncertain in balance, has difficulty in turning and holds the head rigid. In more extreme cases, the patient may resent any movement and such signs as nystagmus and past-pointing may be observed.

A detailed review of the anatomy is tiresome, but some knowledge of structure and physiology is necessary to understand what causes vertigo. The structure is the vestibular system. This includes the labyrinth, the vestibular portion of the VIIIth nerve and central nuclei, which have both afferent and efferent connections with the brain and spinal cord. It is an integral part of the cerebellar mechanism, and its function is the maintenance of balance by orientation of the body in space. This is possible by a constant flow of stimuli from the labyrinth, eyes, muscles and joints, arriving first at the unconscious level and by co-ordinated impulses which are sent to the functioning parts. Normally, these stimuli are in harmony and in accordance with the position of the body. If stimuli which are not in such harmony are received, there is confusion, the clinical evidence of which is vertigo. An example of this is seen in the douching of an ear, causing a convection current in the endolymph of the semicircular canals. This causes a sense of motion which does not coincide with stimuli from other sources and the result is vertigo. Also, "stimulation of the superior lip of the interparietal sulcus evokes violent vertigo." In short, irritation anywhere in the vestibular system giving rise to clashing stimuli causes dizziness. To say that it is an aural vertigo tells nothing, except to imply that the stimulus comes from the ear rather than from a more central spot, because the effect is the same. It may be said then that the mechanism of vertigo, regardless of the basic pathology, lies in a confusion of stimuli causing faulty orientation in space. If we keep this in mind it is easier to understand how different conditions can produce this unpleasant symptom.

In normal persons vertigo may be caused by overstimulation of the labyrinth by motions such as pivoting, swinging and shaking of the head. The original Bárány method of

spinning the patient with the eyes closed used this principle. An additional factor from another source is introduced when a person in a moving vehicle fixes his gaze successively on passing objects, the action of the eyeballs resembling nystagmus. Both sight and the direction of the gaze are aids in orientation in finer degree than impulses from the labyrinth. (A plane pilot flying blind cannot maintain level flight by his sense of balance alone, making necessary the device known as the artificial horizon.) The ocular mechanism is thus associated with vestibular function and plays a part in car, train, airplane and seasickness. Variations in atmospheric pressure from rapid changes in altitude also enter into airplane sickness. These situations are physiological and it is characteristic that nausea and vomiting overshadow the vertigo, because of overflow of stimuli to the vagal nuclei.

A list of the causes of vertigo includes a number of neurological conditions which are varied in pathology and dissimilar in clinical pictures. This seems to make the problem more complex, but it need not if the explanation is sought in terms of localization rather than in the nature of the disease. We return again to the statement that conflicting stimuli arising anywhere in the vestibular system causes vertigo.

The VIIIth nerve is subject to irritation by a variety of conditions, such as arachnoiditis, localized meningitis, degeneration, the effects of trauma and tumors. A neuroma of the VIIIth nerve is a common tumor of the posterior fossa, but other tumors in this location may produce an acute paroxysm by mechanical shifting when the patient lies on the opposite side.

Disease of the cerebellum causes dizziness only because of irritation of the vestibular pathways and their connections. The reaction is in proportion to the acuity of the process. Vascular accidents in the cerebellum, which are usually thrombotic, have a sudden onset. Thrombosis of the posterior inferior cerebellar artery gives a concrete clinical syndrome and deserves special mention. It affects the direct spinocerebellar tract and connections of the vestibular nucleus passing through the inferior cerebellar peduncle. Tumors and local inflammations are less dramatic. Extensive damage to the cerebellum by degeneration, as in cerebellar ataxia, may lead to extreme imbalance but no sense of rotation.

Because the brain stem contains parts of the vestibular system, lesions here may include dizziness among their symptoms.

Degenerative diseases of the spinal tracts, such as the spinocerebellar and posterior column, are said to be a cause but the result is ataxia more than vertigo.

Diffuse diseases like multiple sclerosis and encephalomyelitis are occasional causes. Syphilis may act through the blood vessels or by direct involvement of nerve tissue.

There is a so-called ocular vertigo resulting from diplopia. In extraocular palsies of acute type, in which a person is confronted with a duplication of visual objects, it is natural that he is confused as to which one is in normal relationship with his position. This, however, is probably not the chief cause of the dizziness. Proprioceptive impulses from the extraocular muscles serve a function in spatial orientation, and it is believed that conflict in them produces vertigo.

The fact that irritation in the parietal lobe can cause a violent reaction may explain this symptom in localized lesions, such as tumors and post-traumatic scars. Intracranial conditions of a more diffuse nature probably depend upon alterations of cerebral blood flow. Increased intracranial pressure and postconcussion state may be included here together with cerebral vascular disease.

There is an interesting speculation in respect to migraine and epilepsy. Dizziness sometimes accompanies the headache and is an occasional aura of convulsive disorders. Changes in blood vessel calibre have been advanced as a possible cause of both disorders. There is uncertainty, however, whether vertigo is caused by changes in blood flow in the cerebrum or in the labyrinth itself, where its effect is undoubted.

SUMMARY.

The symptom, vertigo, has been described.

Its mechanism in relation to anatomy and physiology has been given as a conflict of stimuli within the vestibular system resulting in spatial disorientation.

Neurological conditions causing vertigo have been mentioned.

121 University Place.

CHRONIC VASOMOTOR RHINITIS. A CLINICAL INVESTIGATION OF ITS TREATMENT WITH A SCLEROSING AGENT.*†

DR. SAMUEL L. FOX, Baltimore.

Nasal obstruction and its attendant symptomatology constitute the complaint of a large number of patients seeking otolaryngologic aid. In many of these patients there is evidence of some structural deformity of the nose, such as deflections and spurs of the septum, hypertrophied turbinates, or deformities of the anterior nares or posterior choanae, while in others the effects of chronic hyperplastic sinusitis (polypi) account for the obstructive process. In a large portion of the cases, however, no such structural deformity can be uncovered. Instead, there exists only a marked turgescence of the turbinal tissues. In most cases, this turgescence responds well to the sympatheticogenic drugs (adrenalin, ephedrine, neosynephrin, paradrine, etc.) so that the obstruction is temporarily relieved by their prescription.

ETIOLOGY AND PATHOGENESIS.

The exact etiology of this condition, which the author terms "chronic vasomotor rhinitis," may or may not be discovered by otolaryngologic examination. Some cases are definitely the result of allergy; in fact, a large percentage of the cases would probably fall into this category if better means of study were available. Another etiologic factor is sudden change in temperature. The condition is often noted in those who work outdoors and in whom the symptoms are immediately manifest on coming inside a building where the temperature is considerably different. Butchers who work in and out of refrigerated rooms and attendants in air-conditioned buildings are frequent sufferers. Females are more often affected than males, and especially during periods of excitement (menstrual periods, menopause, pregnancy, etc.). The symptom-complex is noted in young as well as old people, though it is more prevalent in the third, fourth and fifth decades of life. Our mode of living seems to be a contributory factor in this etiology, since "highstrung" individuals are frequent sufferers.

The condition first manifests itself by a simple swelling of

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the turbinal tissues — the stage of simple turgesence. During this stage the sympatheticogenic drugs will readily shrink the tissues and reduce the swelling, thus re-establishing a good airway; however, this sympathetic stimulation, like all physiologic stimulations, is followed by a period of sympathetic depression — parasympathetic activity — during which time the turbinates again become turgescent, even more so than ordinarily. This sets up a vicious cycle, since the patient then uses his nasal spray at more frequent intervals. When this fails, the patient seeks a more active sympatheticogenic drug as a spray. If the process is not properly treated during the first stage, the turbinal tissues will undergo hypertrophy, first of the soft tissues and later of the bony structure.

During the second stage, the nasal mucosa and the turbinal tissues become boggy and intumescent. The mucosa and vasculosa are still reactive to the sympatheticogenic drugs, but stronger concentrations and more frequent applications of them are required. Continued use of such nasal sprays eventually lead to paralysis of the smooth muscle tissue of the vasculosa, so that practically no shrinkage is obtained even with the strongest sympathetic-stimulating drugs. Waterlogging of the mucosa follows, and polypoid degeneration ensues. Examination of the nose with the nasopharyngoscope will reveal large, pale mulberry-like hypertrophies of the posterior ends of the lower turbinates in many of these cases.

Further progress of the condition leads to true bony hypertrophy of the turbinates. When this occurs, practically no increase in the nasal airway can be produced by shrinkage of the mucous membrane. Palpation of the turbinates with a probe or cotton applicator reveals actual bony hypertrophy and not simple turgescence.

THERAPY.

Treatment of this condition has taxed the ingenuity of otolaryngologists since the beginning of this specialty of the medical arts. The older textbooks, though not cognizant of the pathology or pathogenesis of the condition, nonetheless recognized its existence. One of the earliest recommended treatments was the use of lunar caustic stick (fused silver nitrate) and chromic acid bead or trichloracetic acid to produce streaks along the lower turbinate. When effectual, marked destruction of the mucosa resulted, since only by the

destruction of the mucosa could the subcutaneous tissue be cicatrized. Next came the recommendation that a drop or two of phenol or of one of the acids be injected under the mucosa at several points along the turbinal length. The aim was to get scarring of the submucosal tissues without affecting the mucous membrane. Instead, however, tissue necrosis resulted, and occasionally large sloughs and ulcers were the outcome.

Actual cauterization of the turbinal mucosa was then recommended, and this method is still popularly in vogue by many otolaryngologists. With this method, specially made nasal cautery tips are heated electrically to a white heat and then several deep trench-like streaks are made along the full length of the inferior turbinates under local anesthesia. This is followed by marked fibrosis, so that the turbinate thereafter is prevented from swelling more than a moderate amount. If too vigorous cauterization is carried out, the nose may thereafter be dry and crust-laden, and for this reason the method has fallen into disrepute. If properly used, the method yields excellent results in properly selected cases.

Diathermy, in its various forms, next was advocated. (For a description of the methods, the reader is referred to the voluminous literature on the subject.) In the author's experience, diathermic results have been but temporary and hence the method has been discarded by him. In fact, the only reasonably lasting results were obtained in cases where severe reactions occurred, and in a number of these cases severe nasal hemorrhages occurred, and the end-results were attended by a dry nose. The only case of otitis media ever seen by the author following any of the methods described followed a severe diathermic reaction, and in this case the otitis media was fulminating. Although the advocates of diathermy proclaim its principal merit lies in the fact that it does not alter the mucous membrane, the author's experiences with it do not bear this out. In every case where sufficient reaction resulted from the diathermy to yield a good result in the shrinkage of the turbinate, there was a marked slough and coagulum of the turbinal surface, and the end-result was not unlike that produced by actual cauterization. Because of the reasons set forth, the author no longer uses diathermy in the nose for reducing the size of the turbinal tissues.

Zinc ionization has been advocated, but this method is fraught with danger. There is definite destruction of the

nasal mucosa following the use of zinc ionization methods, and although increased breathing space may be provided, the physiology of the nose is so altered that ionization is to be condemned.

In 1940, Thacker[1] presented a preliminary report on the use of sclerosing agents by submucosal injection for reducing the size of the turbinal tissues in cases of "chronic nonspecific, nonallergic obstructive rhinitis which shrank well with astringents." He concluded that "the obstructive type of rhinitis which shrinks well and belongs to the nonallergic and nonspecific group is the most satisfactory for treatment. . . . The chronic rhinitis which fails to shrink well is not amenable to treatment with sclerosing agents." In 1942, Thacker[2] published a more detailed study of the results obtained by the injection of sclerosing agents into the turbinates in 121 cases. In 68 of these cases he employed sodium morrhuate, in 28 cases Sylnasol (sodium psylliate) was injected, and monoethanolamine oleate was injected in the remaining 25 patients. Histologic studies were made of the tissues before and after injection. After the use of either of the sclerosing agents there was a profuse fibrosis throughout the tunica propria, a diminution in the amount of mucus in the lumen of the glands without diminution in the actual number of glands, small round cell infiltration and reduction in the size and number of vascular sinuses. There was no gross or microscopic evidence of atrophy of the tissues even after 18 months. All three preparations produced about equal sclerosing effects, but Sylnasol proved to be the drug of choice since it was devoid of allergic reactions and it is less unpleasant if some of the injection escapes into the nasopharynx.

In 1943, the *Bulletin of Practical Ophthalmology*, published by the staff of Greens' Eye Hospital, San Francisco, published an article on the "Use of Sodium Psylliate in the Treatment of Hay Fever, Vasomotor Rhinorrhea and Chronic Rhinitis."[3] Excellent results were reported but no analysis of cases was presented.

More recently, Strauss[4] reported favorable results in a preliminary report of the use of Sylnasol injected into the soft palate and uvula in cases of snoring. The data presented are too meager and inconclusive to draw accurate conclusions.

In 1940, the author began a clinical investigation of the value of sodium psylliate solution (Sylnasol) in cases of nasal

obstruction. More than 200 cases have been treated to date, and the term "chronic vasomotor rhinitis" has been adopted for the classification of these cases, irrespective of their basic etiology. Approximately one-half of these cases had an allergic background. About one-fourth of the cases had undergone nasal surgery within the six months prior to their treatment by Sylnasol. In these cases, structural nasal deformities had been diagnosed and submucous resections, with or without turbinal infraction or partial resection, had been performed. In spite of this, the nasal obstruction persisted, though to a lesser extent. In the remaining cases, there was a varied, perhaps undiscovered, etiology. In all of the cases, prompt shrinkage of the turbinates and the restoration of an adequate nasal airway could be established temporarily by the use of the sympatheticogenic drugs (neosynephrin being the routine agent employed at the treatment table), and the history of the obstruction was of sufficient duration to term the case "chronic."

Early in the series only 0.25 cc. of Sylnasol solution were injected beneath the mucosa of one lower turbinate at the point of greatest turgescence. A week later the same dose was injected beneath the mucosa of the opposite lower turbinate. A few cases reacted adequately to one injection on either side, a larger number required two injections on either side, and the largest number required three injections on either side. It was soon apparent that larger doses could be injected safely, and that both sides could be injected at the same sitting. Now, 0.5 cc. of Sylnasol are injected beneath the mucosa of each lower turbinate at the same sitting and the procedure is repeated in from one to six weeks if necessary. Using this latter method, approximately one-half the cases require only one injection on either side, the remaining requiring a second injection on either side. Only an occasional case requires a third injection on one or both sides, and then usually 0.25 cc. suffice for this injection.

The results obtained in this series of cases have been excellent. In every case there has been a noticeable change in the size of the lower turbinates. In not all of these, however, has the resultant change been entirely adequate. Of the definitely allergic cases, over 60 per cent responded to the treatment. Some of the cases have been observed through two ragweed seasons and are definitely more comfortable. Three cases

were treated by a further injection at the height of the second year's season, and with added benefit. Of the "nonallergic" cases, over 75 per cent benefited from the injections and required no further treatment. The remaining cases were so obstinate that it was thought best to proceed with a galvanocauterization, either alone or preceded by a lateral infraction of the lower turbinates. The author would emphasize, however, that as the dosage used was increased, the percentage of refractory cases decreased. Early in the series only about two-thirds of the cases responded adequately, the remaining one-third requiring galvanocauterization. At the present time, less than 10 per cent of the cases chosen for the sclerosing (injection) therapy fail to respond and require galvanocauterization. In practically every case where the nasal obstruction was relieved, the accompanying symptoms of headache, nasal neuralgia, dry throat, sneezing, etc., were also proportionately relieved. There was a particularly noticeable decrease in the production of mucus and of the dripping of this mucus postnasally into the pharynx.

TABLE 1. STATISTICAL STUDY OF 200 CASES OF "CHRONIC VASOMOTOR RHINITIS" TREATED WITH SUBMUCOSAL INJECTIONS OF SYLNASOL.

Type of Case	Number	Definite Improvement	Little or No Improvement
Cases with definite allergic history	96	58	38
"Nonallergic" cases without structural deformity	53	43	10
"Nonallergic" cases after nasal operation for correction of structural deformity	51	37	14
Totals	200	138	62

TECHNIQUE OF INJECTION THERAPY.

After carefully selecting the case as one suitable for injection therapy, the operator must postpone the therapy until the next visit, as the procedure used to determine the suitability of the case vitiates against its use; namely, the ability of the turbinates to shrink is determined by the use of a sympatheticogenic spray. Once the turbinates are shrunk, it is difficult to inject the solution under the mucosa, as the bony structure of the turbinate is very close to the surface. It is best, then, to make the injection without benefit of shrinkage. As the local anesthetic agent, long cotton pledgets saturated with 0.5 per cent Pontocaine solution are inserted between

the lower turbinates and the septum, since Pontocaine does not produce vasoconstriction. After about five minutes, the pledgets are removed and the Sylnasol solution is injected, using a 2 cc. tuberculin syringe and a 23-gauge steel needle, two inches long. The needle is inserted beneath the mucosa anteriorly and gently pushed back into the turbinate, being careful not to make a counter-puncture. A full 0.5 cc. of the Sylnasol solution are injected as the needle is slowly withdrawn. A small, dry cotton pledget is held in readiness to place against the needle puncture point to control any bleeding after withdrawal of the needle. The opposite lower turbinate is injected in a similar manner at once. The patient almost immediately complains of some retrobulbar discomfort, but this is of short duration — a matter of five or 10 minutes at most — and seldom reaches the proportions of actual pain. The discomfort is equally great after the injection of 0.25 cc. as after 0.5 cc. Occasionally, pain is complained of in the teeth, but this is very transient, too. If any of the solution drops into the pharynx as a result of accidental counter-puncture of the mucosa, the patient will complain of an acrid taste and burning sensation, which may occasionally cause coughing for a few minutes.

Thereafter, the only complaint is nasal obstruction, and this is only slightly relieved by nasal sprays, so that the author does not advise their use unless the patient is very uncomfortable. In from three to five days the reaction begins to subside, and in from seven to ten days actual shrinkage can be observed. This process continues for some five to six weeks. In addition to the shrinkage of the nasal turbinates, there is a definite diminution in the production of nasal mucus, so that many of the patients who formerly complained of "postnasal drip" are markedly relieved of this annoying symptom.

The author does not employ Sylnasol injections into the middle turbinate, as chronic enlargement of the middle turbinate is due to actual bone and soft tissue hypertrophy, and is not relieved by sclerosing agents. Furthermore, injections into the middle turbinate are not without danger, due to the intricate venous communications of this turbinate with the cavernous sinus.

SUMMARY AND CONCLUSIONS.

A clinical investigation was undertaken to determine the

value of sodium psylliate (Sylnasol) as a sclerosing agent in the nose. More than 200 cases were treated in which there was nasal obstruction due to vasomotor paralysis of the vasculosa of the turbinal tissues. The author terms this condition "chronic vasomotor rhinitis," and, depending on the degree of the pathology, classifies the cases into three groups as follows:

1. The mild cases, which require no therapy other than a discontinuance of the chronic use of the sympatheticogenic nasal sprays.

2. The moderately advanced cases, which, nonetheless, respond immediately to the sympatheticogenic drugs and show little or no evidence of actual turbinal hypertrophy. The sclerosing agents find their greatest usefulness in this type of case, and since this group contains the largest number of cases, it is the one most often dealt with in practice.

3. The advanced cases, in which actual turbinal hypertophy accompanies the turgescence. In these cases some surgical procedure will be required, such as lateral infraction and compression of the lower turbinates, removal of the hypertrophied posterior ends of the lower turbinates, galvanocauterization of the lower turbinates, middle turbinectomy (partial or complete), or some combination of these procedures. Few, if any, of these cases respond to sclerosing therapy.

The results of treatment with Sylnasol injections of more than 200 cases in Group 2 proved highly successful. There was not only relief of nasal obstruction but the accompanying symptoms (postnasal drip, headache, nasal neuralgia, dry throat and sneezing) were also relieved. The method bids fair to assuming an important rôle in the treatment of allergic rhinitis.

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1820 Eutaw Place.

VITAMINS AND THE EYE, EAR, NOSE AND THROAT. A REVIEW OF RECENT LITERATURE.

DR. ISAAC H. JONES, DR. HAROLD S. MUCKLESTON, DR. EUGENE R. LEWIS, DR. WALTER P. COVELL and MAJOR LELAND G. HUNNICUTT.

It seems that Vitamins, instead of being a "nine day wonder" have been a nine year wonder. The older otolaryngologists have seen four subjects emerge and have watched them develop from the beginning — focal infection, endocrines, allergy and more recently the vitamins. The sudden intense interest in the vitamins throughout the world, however, very soon applied the available knowledge to clinical work; and an astonishing literature gave the impression that here we had "Cures" for many conditions that had baffled us. To an appreciable extent, this has turned out to be true. However, in reading the available recent literature we have found that most of it concerns purely scientific study — largely chemistry — and perhaps only one-tenth of it has any clinical application. "New" vitamins are no longer appearing every few months as they used to; and even the recent reports of clinical results with the well-known vitamins are relatively few — particularly in diseases of the eye, ear, nose and throat.

One new and sweeping statement has appeared — that of Dunlap and Loken.[1,2] They report curing defective color vision by vitamin A. These reports come from a department of Psychology and lead to conclusions quite different from previously accepted views. As recorded by Jones[3] we have always been taught that the retina is capable of detecting vibrations between 400 million million and 800 million million a second. When only 400 million million strike the eye we see red. When 800 million million vibrations strike the retina, we see violet. When all vibrations between "400" and "800" strike the retina at the same time, we see white. We cannot see anything below red or above violet. All around us, constantly, are vibrations ranging from the lowest to the

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highest; but we are conscious of only a few of them. We do not possess the necessary receptors for the others. If a retina has no receptors for 400 million million vibrations, one simply cannot see red. This has been the accepted concept.

A large number of applicants for flying training who have been disqualified because of defective color vision have sought help from individuals who claim they can improve or restore color vision by a system of eye exercises. In trying to explain the facts to such applicants and to speak in language they understand and remember, it has been our custom to say "If you wish to have good color vision, just go to some revivalist — and be *born again*". In brief, we have always considered that any individual can or cannot see colors, according to the equipment with which he was born.

Now, in sharp contrast, Dunlap and Loken report that they have enabled many patients to pass the standard color-vision tests in which they had previously failed — the Stilling, Ishihara and other tests in routine use. Similarly those who had failed in the test with the colored yarns were made normal. Dunlap and Loken state that they do not know how permanent the cures will prove to be. They state that color-blindness of the so-called "red-blind" type is obviously not the simple "sex-linked Mendelian character" which popular theories have assumed it to be; and that persons whom tests prove to be color-blind but who have not known it, may now reasonably be suspected of not having been color-blind very long. Their treatment consists of "A" in doses of 25,000 units; most cases are cleared up in from three to eight weeks by one dose per day. Giving 50,000 units a day seems to accelerate the cure but upsets some digestive tracts; then they give 25,000 units after breakfast and a similar dose after dinner, and if digestive trouble still results they reduce to one dose a day. They consider that the extensive use of the charts for testing in the present war has brought it sharply to public attention that these tests not only are unfair, but are also unsafe; and that it would actually be safer to discard these color tests altogether. After receiving adequate quantities of "A", about 80% of those who had failed were able to pass the tests. One confusing feature of their report is that the authors regard as

cured the cases which succeed in passing the very tests they condemn.

Color vision is a very important subject. Although we defer to the physiologist, or the ophthalmologist who has delved deeply into the physiology of vision, yet we have not been without practical experience on this subject and so venture to record our own observations. Two of us were concerned with the writing of the Aviation Examination in May, 1917; we were consultants to Gen. Theodore C. Lyster in formulating this "A.G.O.609". This was originally prepared for the Army and later was adopted by the Navy, Marine Corps, Department of Commerce and Civil Aeronautics Authority. The tests for color vision used at the present time were a part of our original examination. One of us has continued to study aviators for the past 26 years; and in the past four years has examined about 4,000 applicants for military service — in the Royal Air Force of Great Britain and of Canada, the Eagle Squadron, the Ferry Command, and, after Pearl Harbor, the Air Forces of our Army, Navy and Marine Corps. The idea never occurred to us that color vision could be restored to those who did not have it. About the usual percentage of applicants failed in our tests of color vision. Without any suggestion from us, no doubt having read or heard that "Vitamin A is good for the eyes", many took "A" and then returned to us for reexamination. Several must have used all of their ration points to purchase cans of carrot juice which they drank in large quantities for weeks. In no instance was there any improvement in color vision.

Again, as to the general subject of vitamin deficiency, some years ago we coined — "No deficiency — no cure". Even if it were true that color vision can be restored by any vitamin, such a result would surely not be expected in any individual unless he has a vitamin deficiency. It is difficult to believe that 80% of those who went to Dunlap and Loken had an "A" deficiency. Most applicants for aviation are alert and intelligent. If dubious about passing visual acuity tests they will memorize the chart, if they get the chance. In such a case a different chart may be used — to their chagrin. This might account for those who passed the color tests in which they had previously failed. It may be that they memorized the color charts. This could have been detected simply by

showing them the charts in a different sequence. This possible explanation of the findings of Dunlap and Loken, however, would not explain the reported improvement in matching colored yarns in the Holmgren test.

We will not attempt to discuss the physiology or the tests of color vision, but will confine our comments to what is known to date about vitamins and retinal function. Much research has been done on this subject. The precise effects of "A" are not yet clear. Morton[4] speaks of the notable advances that were first made and then rapidly confirmed — only to be followed by a difficult phase in which experiments yield apparently contradictory results. It is now realized that the subject is very complicated and the spurious clarity which was induced by too simple a picture has gone altogether. Discrepancies and anomalies are being faced, however, and rapid progress towards a clearer interpretation is to be expected.

The lack of agreement as to what constitutes subclinical "A" deficiency and the uncertainty existing as to the order of the occurrence of the early signs prompted Brenner and Roberts[5] to undertake two studies — one on animals and one on human beings. Six university students, 4 women and 2 men, were placed on a diet which was low in "A". The women were kept on this diet for 7½ months and the men for 4½ months. At intervals measurements were made of dark adaptation, the amount of "A" in the blood, the total and differential leucocyte counts and changes in the structure of the skin and conjunctiva. None of these measurements showed any definite changes. These studies indicate that either the subjects had sufficient storage to withstand the effect of depletion or that the physical signs generally attributed to a lack of "A" are not merely the result of an uncomplicated "A" deficiency. It is interesting to note that a calculation based on the average "A" content of human liver and the assumption that the rate of use is approximately 2,000 units a day indicates that it would take from one to two years for the liver to lose its entire stores of "A" — even if no "A" were taken in the diet. The specificity of night-blindness as an indication of an "A" deficiency has been challenged by the reported curative effect of "B_2" and also "C". Animal experimentation has revealed that the "A" content of the

retina persists even in the absence of stores of "A" in the liver.

In studies with white rats, Brenner, Brookes and Roberts[6] found that on a diet free from "A": (1) 4-weeks old rats lost their total storage of "A" in the liver after one week; (2) the "A" content of the blood declined rapidly at first and then more gradually; (3) the "A" content of the retina decreased up to the 8th week of depletion — and then a plateau was reached; and (4) in the absence of storage of "A" in the liver for several weeks, "A" was still present in the blood and in the eye.

Throughout his many years in India, Wright[7] found that an "A" deficiency is very common in the region of Madras. He has long urged that it is the greatest cause of preventable blindness in South India children. However, it is his considered opinion that an "A" deficiency disease problem in Great Britain or America is unthinkable on diets short of famine diets. Night-blindness in the military services is not merely an "A" problem, and will not be solved by "following the carrot".

The determining of an "A" deficiency itself is not a simple matter. A study of dark adaptation tests in detecting an "A" deficiency was made by Oldham, Roberts, MacLennan and Schlutz.[8] A similar study had been made by Steininger and Roberts who used the biophotometer; they concluded that such a test was not reliable in making a diagnosis of subclinical "A" deficiency. Another instrument, the adaptometer, was designed by Hecht and Shlaer; as this instrument seemed to be an improvement on the biophotometer, it seemed wise to use it in a new survey of this problem. Three groups of children from different socio-economic levels were tested. It was found that the three groups, known to have widely different "A" intake, showed almost identical thresholds of dark adaptation; approximately the same percentage of subjects in each group had subnormal dark adaptation. A slight but significant improvement was found in the children who received 25,000 units each school day for a period of nine weeks, but the conclusion of the study indicated that the adaptometer does not give an accurate indication of the first signs of an "A" deficiency.

Not only does it appear difficult to produce a visual defect

by an "A" deficiency, but Rosenberg[9] has shown that "B_2" in free form plays an important role in retinal function. Light converts "B_2" into a "photo-compound" of unknown structure; this process seems to have a bearing on the stimulation of the optic nerve. The primary "photo-compound" is extremely sensitive. In the absence of oxygen it is destroyed; in the presence of oxygen it is reconverted into "B_2". The mechanism of the "B_2" action in the retina is especially well understood for dim light, since light of short wave length is converted into light of longer (yellow-green) waves by the fluorescent activity of "B_2". The human eye has a maximal sensitivity for greenish light.

In addition to "A", Knapp[10] considers that "D" is also vitally concerned in night-blindness. He fed pups diets low in "D" and calcium, and found changes comparable to those seen in retinitis pigmentosa — scattered areas of pigment, disc pallor, narrowing of the arterioles and posterior cortical cataract. Nine human subjects who had retinitis pigmentosa received "D" and calcium over periods varying between 2 months and 7 years. Night vision improved in all; the discs changed from pale to pink in 5 cases; and the arteriole caliber increased in 3 cases. Similar improvement was noted in 48 myopes with nyctalopia.

Dunlap and Loken have apparently considered only "A" as bearing upon this problem. To Sydenstricker[11] it appears hazardous to be dogmatic in the interpretation of the effects of a single vitamin. The lack of one vitamin has an influence which we cannot yet appraise, on the physiologic functions of others. It is probable that "chain reactions", requiring the synergistic activity of many vitamins, are of prime importance. For example there are indications that "B_2" and "C" must be present in adequate amounts in order that "A" may be properly utilized.

The evidence indicates that at least four vitamins are concerned in the function of the retina.

VITAMINS IN GENERAL.

Our armed forces are perhaps the best fed people in the world.[12] Howe[13] and Brown[14] discuss the rations of the Army and Navy, respectively. The basis of calculation and ration-

ing in the two Services is somewhat different; the two systems, however, accomplish the same end — a well fed armed force.

In discussing the vitamin content of foods, Bing[15] mentions the variations to be found in many natural foods. There is an old fallacy that an egg is an egg and beans are beans; yet there may be tremendous differences in their respective compositions. Pork is richer in "B_1" than other meats. Salmon contains more niacin than cod. The "A" content of milk varies with the season. American cheese contains six times more "B_2" than cream cheese. The darkest carrots may contain twelve times more carotene than pale carrots. Oranges picked while green, contrary to general opinion, do not have a lower "C" content than when picked fully ripe; indeed they may have a somewhat higher content. As to the effect of cooking — it appears that there is no loss of "B_1" in boiling carrots; about one-third of the "B_1" in potatoes is lost in boiling, less in baking. Green peas lose an appreciable amount of "B_1" after simmering for twelve minutes. If a pinch of soda is added the peas are greener, but show an appreciable loss of "B_1". Navy beans lost no "B_1" nor did the addition of a trace of soda decrease the amount of "B_1". The cooking of rolled oats or whole wheat in a double boiler showed no loss of "B_1", but a loss of 14% of "B_1" occurred when either was cooked in the form of bread. There was a loss of 15% of "B_1" in pork as a result of braising and a 57% loss from roasting. Boiled potatoes, cooked spinach and carrots — each furnishes amounts of "B_1" per serving which are comparable to those supplied by cooked cereal or a slice of whole wheat bread. Boiling potatoes for 35 minutes causes no loss of "C". A serving of potatoes provides from 14 to 25 mgms. of "C". Apparently "C" is largely destroyed by mashing potatoes or whipping them. Some day there will be a Cook Book really based on the principles of nutrition. Such a volume will dispel many of our present prejudices. Frying is considered the least desirable method of preparing foods and yet fried meats retain more "B_1". The addition of soda has been objected to on the grounds that it aids in the destruction of "B_1" and of "C". This is true in most products, but in the cooking of dry beans a trace of soda has no deleterious effect and apparently shortens cooking time.

The remarkable book of Price[16] covers his detailed observa-

tions in many parts of the world. In the Foreword, Hooton[17] appreciates and brings out the new concepts in this book. He recognizes that there is nothing new in the observation that savages, or peoples living under primitive conditions, have excellent teeth; nor is it news that most civilized peoples possess wretched teeth which begin to decay almost before they have erupted completely. Indeed this has been a matter of grave concern for more than a generation. It seems to Hooton that we have been extraordinarily stupid in concentrating all of our attention upon finding out why our teeth are so poor, without ever bothering to learn why savages' teeth are so good. He admires Price for being the only person who possessed the scientific horse sense to supplement his knowledge of the probable causes of dental disease with a study of the dietary details which are associated with dental health. Price has found out why primitive men have good teeth and why their teeth go bad when they become civilized; but he has not stopped there — he has gone on to apply his knowledge acquired from savages to the problems of their less intelligent civilized brothers. We must admit that if savages know enough to eat the things which keep their teeth healthy they are more intelligent in dietary matters than we are. One example of the unconscious wisdom of primitive peoples is noted by Price. Special feeding is prescribed for the prospective bride — in some tribes for as long as six to eight months. Price makes clear that the teeth which are the special objects of his study are fair criteria for all tissues of the body. Even the process of thinking is as biologic as is digestion, and embryonic brain defects are as biologic as are club feet. Profuse illustrations contrast the excellence of the teeth and dental arches of primitive peoples with the degenerations seen in those who eat our average modern foods. These observations cover the isolated and modernized Swiss, Gaelics, Eskimos, North American Indians, Melanesians, Polynesians, African tribes, Australian aborigines, Torres Strait Islanders, New Zealand Maoris and Peruvian Indians.

The comprehensive book on the chemistry and physiology of the vitamins by Rosenberg[9] is a valuable contribution. All known vitamins, with the probable exception of "D" are synthesized by plants and then used by them essentially for the same purposes as by man and animals. The difference is that man, unlike the plants, is not provided with means to produce

the vitamins; the only exceptions are "D" and biotin. Those activators which are produced within man and animals and which otherwise conform to the definition for vitamins, are classified as hormones. Vitamins are compounds which are effective in small amounts and occur as traces in cells and body fluids. They do not furnish energy; the amount of vitamins needed is too small to account for even a fraction of the total energy. Actually, minute quantities of vitamins are burned in the organism but the energy set free by this process is infinitesimal. The vitamins furthermore do not act as structural building units. Most vitamins when taken in excessive amounts are excreted unchanged; this indicates that they are utilized neither as suppliers of energy nor as structural building units. The vitamins are, however, essential for the transformation of energy and for the regulation of metabolism. It is not to be supposed that all animals need exactly the same nutritional elements; for example, it has been observed that the cockroach apparently does not need any "A". On the other hand, "C", while apparently needed by all animals, is synthesized by many of them, whereas the primates and the guinea pig are dependent upon an outside source of "C". The physiologic action of vitamins includes their influence on the secretion of hormones and on the metabolism of inorganic compounds. However, Rosenberg considers that no physiologic relation exists between any one vitamin and another. "B_1" is not stored in the organism; the amount actually present is enough to maintain normal life only a few days. A daily intake of "B_1" is therefore necessary. The organism absorbs only as much "B_1" as is needed for the time being; of the excess, some is destroyed and the rest excreted. Even intramuscular injection of "B_1" causes immediate excretion in the urine. "B_1" deficiency can be detected from the amount of the vitamin excreted in the urine; a normal person should excrete 20-80 units per day. Not only is "B_2" a factor in the function of the retina, but the theory has been advanced that it also takes part in an oxidation system in the cornea. Since the cornea is avascular, the cells of the cornea are nourished, according to this theory, by a specific enzyme system containing "B_2". It is thought that during a "B_2" deficiency the body attempts to counteract the lack of oxygen by increased vascularization. An interesting fact about "B_2" is that the phosphorescence of the glow-

worm is caused by "B_2" in combination with a special protein. "D" given in large excess to any experimental animal or to man is toxic. The first sign is digestive disorder, with loss of appetite, vomiting and diarrhea. A considerable loss of weight, inflammation of the kidneys and finally death occur. Excessive doses of "D" cause an increase of the calcium content of the serum; as a result calcification occurs in various organs and tissues, especially in the kidneys, stomach, lungs, heart, blood vessels and bronchi. Unfortunately no exact data for the toxic dose can be given.

Nutrition in the tropics and in the Far East was studied by Van Veen,[18] while living in Java. In China, Japan, India and Java the population is so dense that mere existence is difficult. The people are largely dependent on the produce of their own soil, no matter how unsuitable this may be. Many of the best foodstuffs are sold and the less nutritious kept for home use. Food is almost exclusively vegetable. Other nutritional factors are tradition, habits, religion and ignorance. Meager as the diet often is, its value is further impaired by malaria and intestinal parasitism. Proteins and fats in the tropics are largely of vegetable origin.

In a brief review, Bothman[19] has touched on many abnormal conditions of the eye that can result from a food deficiency. Night-blindness was known in ancient Greece and for this condition Hippocrates prescribed "as big an ox liver as possible, raw and dipped in honey". An "A" deficiency may cause xerophthalmia, keratomalacia, cysts of the lids and injection of the meibomian glands, calcareous deposits in the palpebral conjunctivae, conjunctivitis with lack of luster and wrinkling of the conjunctiva, pigmentation of the conjunctiva, blepharitis and hordeolum, decrease in tears, edema and puffiness of the lids, comedones and night-blindness. The Japanese are a myopic race and live on a diet that is low in "A". It is interesting to note that myopia is not common in American-born Japanese on the average American diet. Corneal inflammation, ulcers and opacities have been ascribed to a lack of the B-Complex. Optic neuritis may be due to "B_1" deficiency. A lack of "B_2" is considered a not infrequent cause of keratitis. "C" is recommended as preoperative treatment for cataract; and occasionally it proves of value in retinal and vitreous hemorrhages.

Post-operative hemorrhage after cataract extraction is one of the more common accidents; its occurrence varies from 1 to 35 percent in available reports. De Voe[20] studied 453 such cases, in the hope that it might be prevented if only the cause could be discovered. He considered seasonal incidence, comparison between ward and private patients, blood pressure, diabetes, intraocular pressure, trauma, pain, the type of operation — and other possible factors including "C" nutrition, capillary fragility and prothrombin in its relation to "K." It has long been known that a serious lack of "C" results in bleeding from many tissues. "C" is needed to maintain the intercellular supporting materials which provide the framework for healing. Man apparently is incapable of synthesizing "C", and there is probably little or no storage in the human body. Daily intake of 100 mg. will maintain a normal individual at the proper level. When more is fed the excretion of "C" rises immediately. Capillary fragility is considered by some investigators to parallel the "C" nutrition. In this connection the existence of "P", which has been thought to influence capillary fragility, has been debated but as yet is not generally accepted. In a total "C" deficiency, failure in healing occurs and is manifested microscopically by a lack of intercellular substance; this substance reappears within 10 days after large doses of "C". Reports of others suggested that there is less likelihood of hemorrhage after an intraocular operation if the patient is saturated with "C". In this large series De Voe concludes that whereas persons with a low "C" content are more apt to have hemorrhage, those in the generally accepted normal range show the usual incidence of hemorrhage; bleeding was encountered in persons with high "C" levels. As to capillary fragility, this test proved to have no value as to whether hemorrhage was apt to occur or not. Prothrombin is now thought to be elaborated only in the liver from derivatives of ingested plants and certain vegetable oils; it is assumed either that "K" is a building stone in the formation of prothrombin or that it is necessary in maintaining hepatic function. Being fat-soluble, it is absorbed only in the presence of bile — because the bile prepares fat for digestion and absorption. Four factors are necessary to maintain a normal prothrombin level: normal bile in the intestinal tract, a diet containing "K" or material from which it can be manufactured, normal absorption, and an

adequately functioning liver. It is now generally felt that there is no use in giving "K" unless there is a deficiency in prothrombin. Hemorrhage occurs only after an 80 percent loss of prothrombin. De Voe concludes that a lack of "K" is not an important cause of hemorrhage after cataract extraction.

Although vitamin nutrition is only a part of the problem in tuberculous patients, Getz and Koerner[21] suggest that it is a major factor. The lack of practical laboratory tests for some of the vitamins limits information about them, but we have such tests for both "A" and "C". These tests were applied to both tuberculous and normal subjects, providing a basis for comparison. It was found that the level of "A" in the plasma of recently diagnosed tuberculous patients was lowered — and this in proportion to the extent of tuberculous involvement. The same was true of "C". Patients with advanced tuberculosis often show profound deficiencies. The nutritional state of a patient can no longer be judged by weight and appearance, for the tests reveal vitamin deficiencies in overweight as well as in underweight patients.

Cowan, Diehl and Baker[22] mention that repeated studies have shown that both animals and man have a decreased resistance to infections of various kinds, when suffering from vitamin deficiencies. Apparently that may be true for each of the better known vitamins. On the other hand, it has not been shown by controlled experiments that the addition of any of the vitamins to an adequate diet produces increased resistance to infection of the upper respiratory tract —notwithstanding the millions of dollars' worth of vitamin preparations which are sold each year for this alleged purpose. Certain students of the University of Minnesota volunteered to participate in a study of this subject because they were particularly susceptible to colds. 427 students were enrolled in the "cold prevention group". A control group of 194 students were supplied with placebo tablets of the same size, shape, appearance and taste as those given to the larger group. This constituted the study of "C". Another group of 347 students were enrolled in the "multiple vitamin study". Another control group for this experiment consisted of 120 students who received placebo tablets. The conclusion reached

is that neither large doses of "C" alone, or large doses of "A", "B_1", "B_2", "C", "D" and niacin have any important effect on the number or severity of infections of the upper respiratory tract — when administered to young adults who presumably are already on a reasonably adequate diet.

Goodale[23] was impressed with the results of giving the B-Complex and "C" to a patient with definite loss of esophageal peristalsis and laryngeal swelling with loss of motion of the left vocal cord. For three months the 66 year old woman had noticed some weakness of her voice and some difficulty in swallowing. She also had had several attacks of sore tongue and puffiness around the lips and eyelids. Examination showed swelling of the left arytenoid, the false cord, and the aryepiglottic fold. The left vocal cord was motionless in the mid-line position. Barium remained in the esophagus when the patient remained in the horizontal position — demonstrating a loss of peristalsis. Biopsy of the left arytenoid showed chronic edema. Her diet had been inadequate and with this in mind she received "B_1", niacin, riboflavin and citrus juices. Rather promptly the puffiness of the eyelids and lips disappeared, the edema of the larynx began to subside, and the left vocal cord began to show better motion. The vitamin intake was then increased and it was observed that the swelling in the larynx further subsided. She ceased to have difficulty in swallowing. It would seem that this case was one of intermittent obstruction of the esophagus with weakening of its muscular walls. Recovery was attributed to correction of the vitamin deficiency.

Grouped in one volume[24] one may find many valuable contributions to this subject of vitamins in general.

"A".

While "A" is not found in the urine of the healthy adult, certain abnormal conditions were found by Tomaszewski[25] to lead to its excretion by the kidneys. Among these pathologic states are uremia, nephritis in different stages, carcinoma and pneumonia in the period of high fever.

A study of the level of "A" in the blood of rats by Josephs[26,27] included a determination of the amount of "A" in

the liver. His effort was to find out how the amount in the liver would affect the amount in the blood. He noted that when the "A" was exhausted in the liver and the serum, signs of an "A" deficiency — i.e., a ring of redness about the eyes, loss of appetite, snuffles and keratomalacia — did not appear even after the lapse of three or four weeks.

Since the principal storage center for "A" in the rat is the liver, Treichler, Kemmerer and Fraps[28] studied the livers of rats which had received different preparations containing "A". Cod liver oil was found to be the most efficient for building up the stores of "A" in the liver; carotene dissolved in cottonseed oil was only 59% as effective.

The reason for carotene being less effective as a source of "A" when combined with pure oil than when combined with crude oils was investigated by Quackenbush, Cox and Steenbock.[29] They found that daily supplements of carotene in ethyl linolate failed to produce growth in young "A" deficient rats. When a distillate of soy bean oil was added, the carotene was effective and growth resulted. The protective factor proved to be tocopherol.

B COMPLEX.

In reviewing the water-soluble vitamins, Elvehjem[30] discusses nine factors of the B Complex which have been isolated in their crystalline forms; however there are at least a dozen vitamins of varied chemical structures included in the B Complex. Other water-soluble vitamins are "C" and "P", or citrin. For every day nutrition common foods are the best source of all water-soluble vitamins. Any one food may show considerable variation in the amount of these vitamins; different tablets and capsules on the market are misleading because of their tremendous variations. The composition of 119 vitamin products, representing 32 different companies, were compared for vitamin content. While these products may be useful in the treatment of specific vitamin deficiencies there is no greater security in them than in the proper combination of natural foods. Thiamine, riboflavin, niacin, pyridoxine and pantothenic acid are five of the nine vitamins which have been prepared in crystalline form. The remaining are choline, biotin, inosital and para-aminobenzoic acid. Ten years ago Best demonstrated the importance of choline

in the prevention of fatty livers in depancreatized dogs. Choline may stimulate phospholipid formation, be a factor in the production of acetylcholine or supply necessary methyl groups. It is an essential factor for lactation in adult rats and prevents paralysis in suckling rats. As yet no clinical use for choline has been found. Meat, eggs, vegetables and cereals are good sources of choline. Biotin is a very stable compound; however in pure form it is slightly labile to alkali. Animals on diets rich in egg white develop a dermatitis. There is a biotin inactivating factor in egg white — avidin. If the available biotin is tied up with this factor then so-called "egg white injury" results. This is manifested by the rat in biotin deficiency of 10% levels of egg white, as "spectacled eye", which progresses to generalized alopecia and later to spasticity and death. The relationship between biotin deficiency and human ills is unknown; when biotin in pure form becomes generally available a clinical application may be found. Liver, kidney, yeast and egg yolk are the best sources of biotin. Inositol is extremely stable; it occurs in various tissues and cells of the body (including muscle, brain, erythrocytes and eye) — and in plants — as "phytin". Cereal brans and seeds are good sources of inositol. Para-aminobenzoic acid may finally prove of some value in restoration of hair pigments. A lack of it in the diet of rats produces graying of the fur even though the diet is adequate in pantothenic acid. It is a factor in the growth of chicks and in pregnancy and lactation of rats.

A vast amount of scientific work on the vitamins is constantly being carried on. For example in an exhaustive survey György[31] mentions, in passing, that these 480 research studies cover only a single year and are restricted to the water-soluble vitamins — he calls his survey merely a "limited cross-section". Apparently the best test for "B_1" deficiency is to determine its amount in the urine. If there is a small amount in the urine, we can be reasonably sure that there is a small amount in the body. Urinary excretion of "B_1" is diminished in a person deficient in "B_1". The minimum on which man can exist is 0.6 mg. of "B_1" daily; to function efficiently he needs twice this amount; and for safety he should receive about 2 mg. This upper limit is seldom reached in the American diet. Liver, and to a less extent, kidney and yeast are rich sources of niacin. Cooking of meat

destroys very little of the niacin. The output of "B_1" in the urine reflects only the immediate intake in the diet; consequently it is not a reliable measure of long-standing deficiency of "B_1". The daily requirement of "B_2" for an adult is approximately 3 mg. As to the other factors of the B Complex, pyridoxin, pantothenic acid, choline and biotin — no definite information is as yet available regarding the roles that they play in the health of the human being.

New information as to the need of an adequate supply of B Complex by the teeth and oral structures is provided by the careful experiments of Becks and Morgan.[32] They conducted experiments in deficiencies of various factors of the B Complex for a period of five years. Such a study of rats and dogs gave them the opportunity to study the conditions of the tissues by deficiencies of the filtrate fraction and of niacin. The "Filtrate fraction" refers to all members of the B Complex that are not adsorbed on fuller's earth. The members that are adsorbed by the fuller's earth are chiefly thiamin, riboflavin and pyridoxin. Certain dogs were used as controls; other dogs received no filtrate fraction; others no niacin; and others were deprived of both the filtrate fraction and niacin. The results of the experiment showed that the absence of the filtrate fraction and niacin led to severe gingival inflammation and infection of the underlying osseous structure. The clinical as well as the histologic aspects resembled the paradentosis frequently observed in human beings. Deficiencies of only the filtrate fraction led to severe malformation and resorption of roots, as well as resorption of paradental bone structure resulting in varying degrees of osteoporosis; deficiencies in only niacin led to gingival disturbances; the black tongue of niacin deficiency may represent an exaggerated form of the changes observed in these gums.

Paralysis agitans and its therapy always present a difficult problem. The earlier studies of Jolliffe, Spies, Bean and others were followed by the work of Loughlin, Myersburg and Wortis.[33] They divided twenty-two patients into two groups, of twelve and ten respectively. The former received daily intravenous doses of "B_1", "B_2", niacin and pyridoxine. To the latter was given 5 cc. of a normal saline, daily. No objective change was noted in the vitamin treated group, except that three men showed a growth of fine hair in scalps par-

tially bald, and one woman said that her hair had ceased to fall out. There was no improvement in the paralysis agitans.

In a study of the influence of a B Complex deficiency on wound healing by Holden and Crile,[34] adult albino rats were used. Their conclusion is that a deficiency in the B Complex has little effect on wound healing. The slight lessening of the tensile strength of the wound seemed to be an effect of the loss of weight rather than of the B Complex deficiency.

"B_1".

To investigate the relation of "B_1" deficiency to optic neuritis, Raman and Abbu[35] studied 34 patients, including 15 with beriberi, who showed edema and peripheral neuritis; 2 cases of toxic peripheral neuritis with tachycardia; 4 cases in which peripheral neuritis was the only symptom; 10 cases of pellagra; 1 case of beriberi with pellagra; and 2 cases of diabetes mellitus. In 20 of these 34 cases there was evidence of involvement of the optic nerve; 16 showed concentric contraction of the visual fields; 2 showed primary atrophy. A diet rich in B Complex fortified with "B_1" resulted, in 5 cases, in an enlargement of the visual fields.

Migraine is considered by Palmer[36] to have three phases: 1. The stage of toxic accumulation; 2. The resulting imbalance of the sympathetic nervous system leading to dilatation of the cerebral blood-vessels; 3. The stage of elimination and recovery. Because "B_1" is regarded as important in normal tissue metabolism, a study was undertaken of the effects of giving "B_1" to a number of cases of severe migraine. Forty patients had careful physical and neurologic examinations and also routine electroencephalographic study. The patients received B Complex orally and also daily injections of 30 to 100 mg. of "B_1" for one or two months. Forty-eight patients received this intensive therapy for periods of 7 mos. to 1 year. Twenty-three of the forty-eight patients were completely relieved of migraine attacks; fourteen others can be classified at this time as responding to treatment, in that they experienced improvement in reduced frequency and severity of headaches. As to acute attacks, in more than 150 instances

(70 per cent) the attack was terminated within one to three hours.

A study of "B_1" deficiency in swine by Wintrobe and associates[37] showed that the most obvious symptoms were anorexia, vomiting, dyspnoea, cyanosis and marked weakness. Cardiac failure appeared suddenly and, unless "B_1" was given to the pigs death occurred promptly. The important finding of this experiment is that there were no neurologic symptoms; and even in animals in which "B_1" deficiency was of long duration, no degenerative changes were found in nervous tissues.

In a study of gastric secretion and the motor activities of the stomach, Wood, Splatt and Maxwell[38] selected 36 patients who had shown absence or subnormal amount of hydrochloric acid in the stomach. Each patient received daily injections of 1,000 units of "B_1" for ten consecutive days. After a careful tabulation it was concluded that "B_1" has no appreciable effect on gastric secretion in man; that "B_1" hastens the emptying time of the stomach of those persons whose gastric emptying time is habitually much longer than normal, but does not influence the rate of evacuation of the stomach of those whose gastric emptying time is normal or excessively rapid.

"B_2".

On depriving rats of "B_2" Calder[39] noted a significant elevation of blood pressure. Some consider that certain types of hypertension may be of metabolic origin; it is thought that the high pressure is caused by diminished oxidative activity of the kidney. In these experiments deprivation of the entire B Complex was followed by a slight fall in blood pressure. Deprivation of only the B_2 Complex was followed by a significant and persistent rise in pressure — which could be reversed by restoring these vitamins to the diet.

NIACIN.

A report in the Archives of Ophthalmology by von Grolman and Angel[40] is of special interest. As the ocular fundus lends itself so well to the study of peripheral vascular changes, the authors studied the effect of niacin on the eye. They found that only 3 out of 20 patients showed intolerance to niacin;

2 showed bradycardia and 1 showed extreme intolerance, manifested by sweating, tremors, gastric symptoms, bradycardia and hypertension.

Molitor[41] finds that niacin is the only vitamin which produces a drug-like effect in those who have no vitamin deficiency; it causes transitory peripheral dilatation. At present it appears that there is no real danger in overdoses of vitamins. Daily feeding with hundreds of times the maintenance dose of many vitamins over the entire life span of rats fails to produce toxic effects. However, prolonged administration of "B_1" in rats has, in the third generation, produced loss of the maternal instinct. "B_1" in moderate daily doses counteracts the toxic effects of thyroxin given daily to animals.

Handler and Dann[42] studied dehydration and electrolytic imbalance in dogs whose diets were deficient in niacin. Subcutaneous injections of physiologic salines to dogs with the deficiency prolonged their lives as much as 180 days and alleviated some of the symptoms of the deficiency. Decreased blood chloride and glucose, and renal impairment suggested dysfunction of the adrenal cortex. The relationship of this dysfunction to niacin deficiency is not clear, because dogs which received saline injections did not die with the typical black tongue.

"C".

That a moderate "C" deficiency — subclinical scurvy — is commonly encountered in medical practice, is brought to our attention by Rinehart and Greenberg.[43] General symptoms of lassitude, fatigability, anorexia and "rheumatic" pains are frequent in "C" deficiency. Gingivitis and gingivo-stomatitis occur commonly enough to warrant the suspicion of a lack of "C". A "C" deficiency should be suspected as a major or contributory factor in cases of unexplained bleeding. The simplest, most direct method of detecting subclinical "C" deficiency is to determine the amount of "C" in fasting plasma. The concentration of "C" in the blood ranges from 0 to 1.3 mg. per cent. This latter value reflects a state of saturation. If "C" is administered, the blood plasma concen tration rises above this level and "C" is excreted in the urine. A plasma concentration of 1.3 mg. per cent represents the usual renal threshold. Naturally if the plasma "C" value is near the saturation level, "C" deficiency does not exist; if,

on the other hand, the plasma "C" is low, it does not necessarily indicate that the individual has a "C" deficiency. The determination of the tissue reserve of "C" is simple. If an individual with reasonable concentration receives a large test dose of "C", the concentration of "C" in the blood plasma will rise, reaching a peak usually in 2½ to 3 hours, and a portion of the "C" will be excreted in the urine. If, on the other hand, the tissues are severely depleted, there will be only a slight rise in the blood plasma "C" concentration and none will be excreted. In most cases in which the fasting plasma "C" level was below 0.1 mg. per cent, demonstrable improvement followed the administration of "C". Concentrations in this range afford strong presumptive evidence of subclinical scurvy.

As one method of detecting a lack of "C", Kruse[44] recommends simple inspection of the gums and also the use of the biomicroscope. Of 49 persons in a low-income group showing lesions of the gums, all showed gross or microscopic lesions of the gums characteristic of a lack of "C". Capillaries become engorged and the gum becomes red; then, as stasis occurs, the gums become swollen — (the usual sequence in any congestion). Infection may then occur, with resulting ulceration and bleeding. As this process becomes chronic one may observe atrophic changes — pitting and recession of the gums. In this series of 49 adults blood plasma tests were made in 42; 43% showed a subnormal amount of "C"; 25 of the group received "C"; the gum lesions cleared up in 2; the others showed varying degrees of improvement. The striking feature in this series was the long time required for complete recovery. Those patients who received "A" and niacinamide showed no improvement of the gums.

The remarkable findings by Chamelin and Funk[45] that injections of whole liver reduced the toxicity of sulfanilamide and diethylstilbestrol in rats are discussed by Pelner.[46] His observations indicate that the beneficial action is due to the high content of "C" in the liver. It has long been known that next to the adrenal glands the liver contains more "C" than all other animal tissues. Pelner has noted a reduction in the sensitivity of patients to large doses of the salicylates and the sulfonamides by concomitant administration of "C" in large doses. Three patients were very sensitive to diethyl-

stilbestrol. "C" was given, by intravenous injection and by mouth, with each tablet of diethylstilbestrol — which they then were able to tolerate.

In experiments on guinea-pigs, Bartlett, Jones and Ryan[47,48] found the tensile strength of wounds much less in animals low in "C" than in those with a high "C" content. They found that this is also true in human beings.

Microscopic sections of the bronchioles of the rabbit's lung were used by Ruskin[49] to study the influence of "C" on the antihistamine action of different drugs — such as epinephrine, benzedrine, ephedrine and calcium. Benzedrine ascorbate produced a rapid recovery in the histamine contracted bronchiole, while benzedrine sulfate prolonged histamine contraction. Epinephrine ascorbate produced prompt recovery from the histamine effect, while epinephrine hydrochloride produced moderate dilatation of the bronchioles. Ephedrine ascorbate produced prompt recovery; ephedrine hydrochloride further constricted the bronchioles; and ephedrine sulfate slightly inhibited recovery from the histamine effect. Calcium ascorbate produced very prompt recovery, while "C" alone showed less marked histamine antagonism.

Working in a native settlement in Angola, West Africa, Strangway[50] encountered many tropical diseases, some offering unusual problems. "Utue unene" causes hemorrhages from the mucous membranes of almost any part of the body. Treatment with orange or lemon juice had met with only partial success, until very large quantities were given— ten to fifteen quarts in a period of a week. Strangway regards "utue unene" as acute scurvy.

"D".

Five groups of forty children each were observed by McBeath and Verlin[51] under conditions as nearly identical as possible during one school year. There were no changes in the diet of any of the children except the addition of chocolate syrup containing "D" to the milk of four of the groups. The control group that received no "D", showed 4.5 new carious teeth per child. In the group receiving a daily supplement of 400 "D" units from cod liver oil, there were 2.48 new carious teeth per child. The maximal effects observed in this

study (a reduction to 1.65 new carious tooth surfaces) were shown in the group receiving a daily supplement of cod liver oil — 800 units of "D" per child. This result was not attained by the use of even larger amounts of viosterol. Cod liver oil gave far better results.

Sufferers from vernal catarrh and ophthalmologists who have to deal with the condition will welcome the hope of remedy held out by Knapp.[52] The problem is a double one — allergy plus avitaminosis. In a series of 47 cases, 43 received "D" in form of viosterol, and calcium in addition. All but one showed subjective and objective improvement. As a rule, good patients failed to improve until six weeks had elapsed. Sixty results were noted in seven to fourteen days but several drops of viosterol were given daily; in obstinate cases the amount was increased to 200 drops.

"K".

The value of synthetic "K" in cases with a tendency to hemorrhage was studied by Townsend and Mills.[53] Their material was gathered from the wards of the Montreal General Hospital. They divide their cases into three groups: (1) Those in which prothrombin disturbance is associated with obstructive jaundice; (2) Those with moderate to severe liver damage, which show prothrombin disturbance; (3) Those showing neither jaundice nor prothrombin disturbance, but a tendency to bleeding. The prothrombin time was determined by Quick's method. In the first group the prothrombin time was restored to normal prior to operation, by courses of injections lasting from three to eight days. From the results in the second group the authors determined that the administration of "K" and synthetic compounds is effective in greater or less degree according to the amount of liver damage. The third group included many and varying cases — hemophilia, nasal hemorrhage, recurrent bleeding into the vitreous, post-operative tonsillar hemorrhage and leukemia. In these conditions the bleeding is unrelated to a prothrombin disturbance and the use of "K" or synthetic compounds is not indicated.

It is because of its value in shortening the coagulation time and in lessening or arresting hemorrhage that "K" has been

of especial interest. The evidence indicates that such benefit results only in patients whose blood exhibits a low prothrombin content. There are certain conditions, however, such as thrombosis and embolism, in which it is desirable to lengthen rather than to shorten coagulation time. Townsend and Mills[54] worked with a substance called synthetic dicoumarin. This is identical with the hemorrhagic agent of spoiled sweet clover. They found that in selected cases they could prolong clotting time. The prolongation thus effected could later be nullified and the clotting time returned to normal by giving "K". They are careful to state, however, that in 8 to 10 days after the withdrawal of dicoumarin the blood can return to normal, even without the use of "K".

Acid formation from sugar in the saliva appears to be an important factor in producing dental caries. With this in mind, Fosdick, Fancher and Calandra[55] searched for a non-toxic substance that would inhibit the acid formation sufficiently so that the saliva could neutralize the acid present. They found that synthetic "K" does just this. They first found that "K" inhibits acid formation in vitro. The "K" has no effect on bacterial growth in the concentrations used, so the inhibition is not caused by any antiseptic properties. They consider it probable that if synthetic "K" were incorporated in sugar candy or chewing gum it would inhibit dental caries. It is interesting to note that "K" is probably one of the substances removed from sugar-cane juice during the refining of sugar.

VITAMINS AND THE "RAW MATERIALS".

Vitamins are activators. In recent years vitamins have so filled the center of the stage as to crowd into the background the essential raw materials which constitute the body. These raw materials are constantly suffering wear and tear, requiring replacement. Activators — whether they be vitamins from without or endocrines from within the body — provide no raw materials. The vitamins and endocrines are valueless except as activators. Raw materials and their products in the body are also valueless without the activators.

Ideal foods provide adequate activators as well as raw materials. An official slogan adopted by our country is "Eat the Basic 7 Every Day".[56] Group 1 — green and yellow vege-

tables — some raw and some cooked, frozen or canned; Group 2 — oranges, tomatoes, grapefruit — or raw cabbage, or salad greens; Group 3 — potatoes and other vegetables and fruits — raw, dried, cooked, frozen or canned; Group 4 — milk and milk products — fluid, evaporated, dried milk or cheese; Group 5 — meat, poultry, fish or eggs — or dried beans, peas, nuts or peanut butter; Group 6 — bread, flour and cereal — natural whole grain, or enriched; Group 7 — butter and margarine (fortified with "A").

A number of articles serve to revive interest in the proteins, carbohydrates, fats and minerals. Protein requirements in normal nutrition are explained by Bell.[57] Protein plays a fundamental role in that it supplies the necessary amino-acids for growth and repair of tissues, and for the manufacture of certain secretions, antigens, hormones, antibodies and enzymes; it furnishes energy incidentally to its transformation into carbohydrate; and it is concerned with transport of fats and other lipoids in the body. Protein forms a considerable part of the non-skeletal structure of the body. When sufficient calories are provided by the fats and carbohydrates, muscular activity has little effect on the protein requirement; only when the calories are inadequate is protein called upon to supply the deficiency. During fasting, most of the energy required for muscular work is derived from fats, only 3 per cent being obtained by the breaking down of protein. The old-fashioned notion of a high protein diet being essential for heavy work is fallacious.

In discussing the fats, Sinclair[58] reminds us that they are a much richer source of energy than either carbohydrates or proteins. For the average adult, about one-third of the calories seems to be a suitable proportion for the fats to supply. The harder the work, the greater is the energy requirement and the greater is the need for more highly concentrated fat diets. Since "A", "D" and "E" are fat-soluble, under natural conditions they occur in association with fats. However, many common foods such as pork fat and vegetable oils contain very small amounts of these vitamins. It is well recognized that the diet, especially for growing children, should be supplemented with fish liver oils in order to insure adequate intake of "D". Rigorous restriction of foods containing animal fats from the diet of persons with xanthoma

(cholesterol accumulations) has in some instances resulted in definite clinical improvement. The prejudice of many people against the margarines as substitutes for butter is based on trivial differences in flavor. In terms of calories, the edible fats on the market are all the same. Being cheaper to produce than animal fats, the processed vegetable fats give better value for the money.

As to the diet during pregnancy, Watson[59] stresses that the fetus is essentially parasitic. It takes what it needs from the mother's blood stream and even draws upon the organic stores of the mother. Nature has decreed that the imperious demands of the unborn child be supplied regardless of the cost to the mother in minerals, tissue reserves and vitamins. The fate of the offspring is dependent upon the nutritional state of the mother before and during pregnancy. One of the common defects is a faulty supply of minerals. During pregnancy the mother needs more calcium, phosphorus, iron and iodine. It is essential that the mother receive sufficient calcium so that she will not have to sacrifice the calcium of her bones and teeth in the interests of the developing fetus. A favorable calcium balance may be assured by giving one quart of milk a day in addition to other calcium-bearing foods such as cheese, fruits and vegetables. Phosphorus, being more widely distributed in foods than calcium, is less apt to be lacking. The mother may become anemic unless she ingests iron-rich foods or iron in some other form, such as iron and ammonium citrate or ferrous sulphate. Sources of food-iron are liver, kidney, lean meat, eggs, whole grain and lightly milled cereals, vegetables, fruits and molasses. Cretinism may be avoided by an adequate intake of iodine — from sea-water foods, fish-liver oils or iodine itself in the form of iodized salt. While the commonest deficiency is probably that of "D", the demand for all the vitamins is increased during pregnancy. The best sources of "A" are milk, cream, butter, cheese, egg yolk, green leafy vegetables, yellow vegetables, liver and fish-liver oils. For "B_1" the mother should receive whole grain and lightly milled cereals, vegetables, fruit and egg yolk; in addition she may need to augment the diet with brewer's yeast and wheat germ. The diet should supply sufficient "B_2" and "C"; but it is particularly important to supply an adequate amount of "D" — so essential for the utilization of calcium and phosphorus to insure calcification of the bones

and teeth of the fetus. Ordinary foodstuffs are notoriously poor in "D"; therefore, the pregnant woman should receive cod liver oil, or its equivalent, in her daily diet. A lack of sunshine increases the need for "D". While no relationship has been established between abortions and a deficiency of "E", women with histories of repeated spontaneous abortions have prolonged later pregnancy to full term, while taking concentrated preparations of "E" in wheat germ oil. Except under unusual circumstances, a diet which is adequate in other respects contains enough "E" to meet all ordinary requirements. Of all members of the population the pregnant woman deserves the greatest nutritional protection.

The nutritional requirements of the mother are discussed by Rabinowitch.[60] Premature births and still-births are common among under-nourished women; and when the fetuses of such women reach maturity and are born alive, they are usually underweight and apt to die within the first ten days of life. Among those that survive for longer periods their chances of living long, unless they are artificially fed, are again greatly reduced because of the diminished capacity of the undernourished mother to produce milk. Of course food is not the only factor which governs the physical fitness and survival of the new-born infant; heredity plays its part — the capacity to grow is inherent in protoplasm independent of the food supply. If the mother has had a poor food supply during the intra-uterine life of the child, it is difficult to make up for this lack after birth. A child whose mother suffered from osteomalacia during the pregnancy is likely to develop rickets, regardless of satisfactory diet of the mother during lactation and of supplementing the infant's diet by all essential food elements. A guinea pig runs about and nibbles green leaves soon after it is born; but the new-born human infant is helpless and is entirely dependent on the mother for its food supply. The proper time to prevent deficiency is not after the child is born, but soon after the onset of pregnancy.

The bearing of nutrition upon resistance to disease is presented by Robertson and Tisdall.[61] There is evidence that a lack of almost any one of the 32 food elements essential for animal nutrition will result in lowered resistance to infection. If a patient is suffering from any disease that lasts more than a week special attention must be given to nutrition. This is

frequently neglected. The presence of infection increases the need of the body for many of the food elements; for example, it has been shown that the amount of "A" and "B" in the blood is markedly lowered during infection; and there is evidence of an increased need for "B_1".

A summary of the values and failings of common foods is given by Newburgh.[62] Milk, vegetables and fruits contain all the dietary essentials except calories. Meat, poultry, fish and eggs are valuable for flavor and protein — but they do not furnish sufficient calcium and vitamins. Whole wheat is an excellent source of calories and also contains a considerable amount of the B Complex. Fats are the best sources of calories and add flavor; however, with the exception of butter, fats have no other value.

In discussing proper feeding Mellanby[63] warns us that, although the sense of hunger or repletion is a good safeguard in determining the amount of food to be eaten, instinct is a poor guide to the choice of which foods to eat. The modern mother ought to know that breast-feeding is much better for herself and for her infant than any form of artificial feeding; the death rate among artificially fed infants is much higher than among breast-fed. Mothers would also be saved much trouble and anxiety if they gave their infants a teaspoonful of cod liver oil — even those that are breast-fed. All breeders of dogs and poultry know this trick and how much loss it saves them; it is high time that all mothers knew and practiced it. Right up to the end of adolescence the daily milk intake should be at least one pint — two pints would be even better. The bones especially need an abundance of calcium and if they do not get it become imperfectly formed; also the teeth are badly formed and more liable to decay. The teeth of civilized man are probably his greatest curse; milk, egg-yolk and cod liver oil will help to produce good teeth and to prevent decay. Too much bread and other cereal foods and sugar hasten the onset of decay. Most vitamins are resistant to reasonable heat — therefore the cooking of food does not spoil its vitamin content. Exceptions to this are fruits and vegetables which on cooking lose, in most cases, their "C". Potatoes are excellent food and have many advantages over cereals; so that they can well be used as a substitute for bread, especially in the case of children. In a small island in the

South Atlantic the inhabitants have had no bread or cereal; all the infants were breast-fed for a long period, and the main articles of diet were milk, mutton, fish, eggs and potatoes. There was no rheumatism, arthritis or rickets on this island; the teeth were almost free from decay; and it is reported that there has never been a death in childbirth. Similar conditions used to hold in the Western Hebrides and Labrador, but the advance of so-called civilization and especially the easier access to our own articles of diet, including white flour and sugar, have changed all this. The farther away people are from a "st e" — that is to say the more they have to depend on the natural produce of the sea and land — the better teeth they have.

Twenty years ago, as pointed out by Orr,[64] more than half the children in poorer districts in Northern Europe suffered from rickets, many of them so badly that their legs were twisted out of shape. We have learned to give milk to children because it is the richest source of calcium. We give cod liver oil because it is rich in "D". When this is done rickets disappears. The lack of calcium and "D" not only causes rickets but it impairs growth. Today children leaving school in Great Britain are about two inches taller than their parents were at the same age.

McCollum[65] states that benefits resulting from sanitation — the prevention of epidemics and the control of the most devastating diseases of childhood, between 1911 and 1935 — increased the average duration of life on this continent by nearly 14 years. Better nutrition promises another great achievement by increasing the efficiency and decreasing the burden of ill-health of the population. Adequate diet for the promotion of health is made up of a number of "little things." From the proteins we derive 9 simple substances called amino-acids — none of which we can do without. We must have at least 13 inorganic or mineral elements; and we must have adequate amounts of the vitamins. Iodine deficiency is widespread in many countries; this can be overcome by giving some form of iodine such as iodized salt. Many are not taking enough calcium and many do not get enough of certain vitamins, because their diets are not well chosen. "A" is closely associated with the health of the mucous membrane which should be an effective barrier against bacteria. Many lack

"B_1". The health of blood vessels is dependent upon an adequate supply of "C". Damage resembling arterio-sclerosis seems to result from bacterial poisons in persons who lack "C". There are three ways in which tooth structure may be influenced by diet. The organ which forms the enamel is dependent on "A". If a deficiency of "A" occurs in the infant or child whose teeth are still forming, the enamel will contain pits and fissures — potential food traps where particles of carbohydrate food lodge and ferment, with the formation of acids injurious to the enamel. If the diet contains an abundance of "A" but not enough "D", the content of phosphate in the blood will fall to so low a level that calcium phosphate, the substance of enamel, cannot be laid down and poor enamel will result. The dentin-forming organ is especially dependent upon an abundance of "C"; if an infant or child whose teeth are forming does not get enough "C" the dentin will be badly affected and the future health of the teeth will be impaired. If our present knowledge of nutrition could be applied to the entire population there is no doubt that the ravages of tooth decay could be reduced tremendously. It cannot be claimed that adequate nutrition is a panacea for all human ills; however, there is no doubt that malnutrition is widespread and of a severity which is reflected in many people in ill health. That it is not necessary to take vitamins in pills was demonstrated by Samson, David and the original Marathon runner; similarly Venus de Milo, Aspasia and Cleopatra derived their charm as the others derived their strength, from natural foods only. It was 20 years ago that McCollum coined a slogan: "Eat what you want, after you have eaten what you should."

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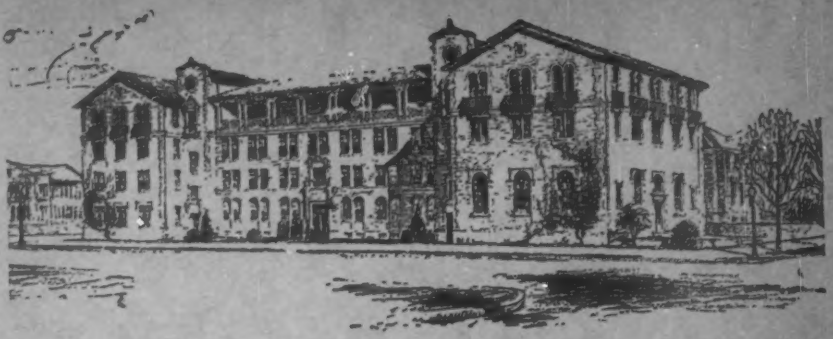
AMERICAN BOARD OF OTOLARYNGOLOGY.

The next examination of the American Board of Otolaryngology will be held in New York City at the Waldorf-Astoria Hotel, June 1-2-3-4, 1944.

The meeting that was tentatively scheduled to be held in Los Angeles in February, 1944, has been canceled.



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